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Edwin Sequeira

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June 21, 2000

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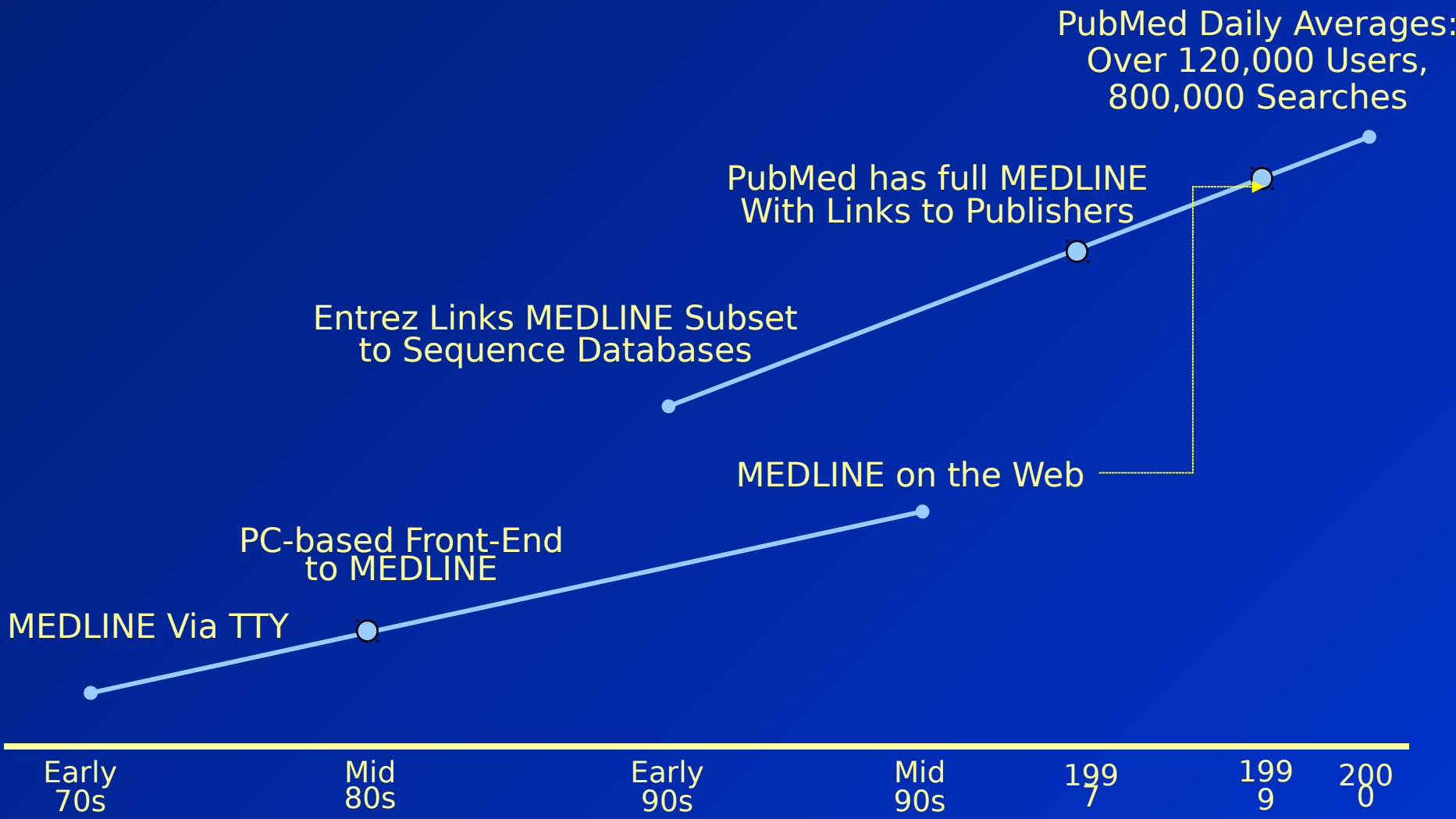

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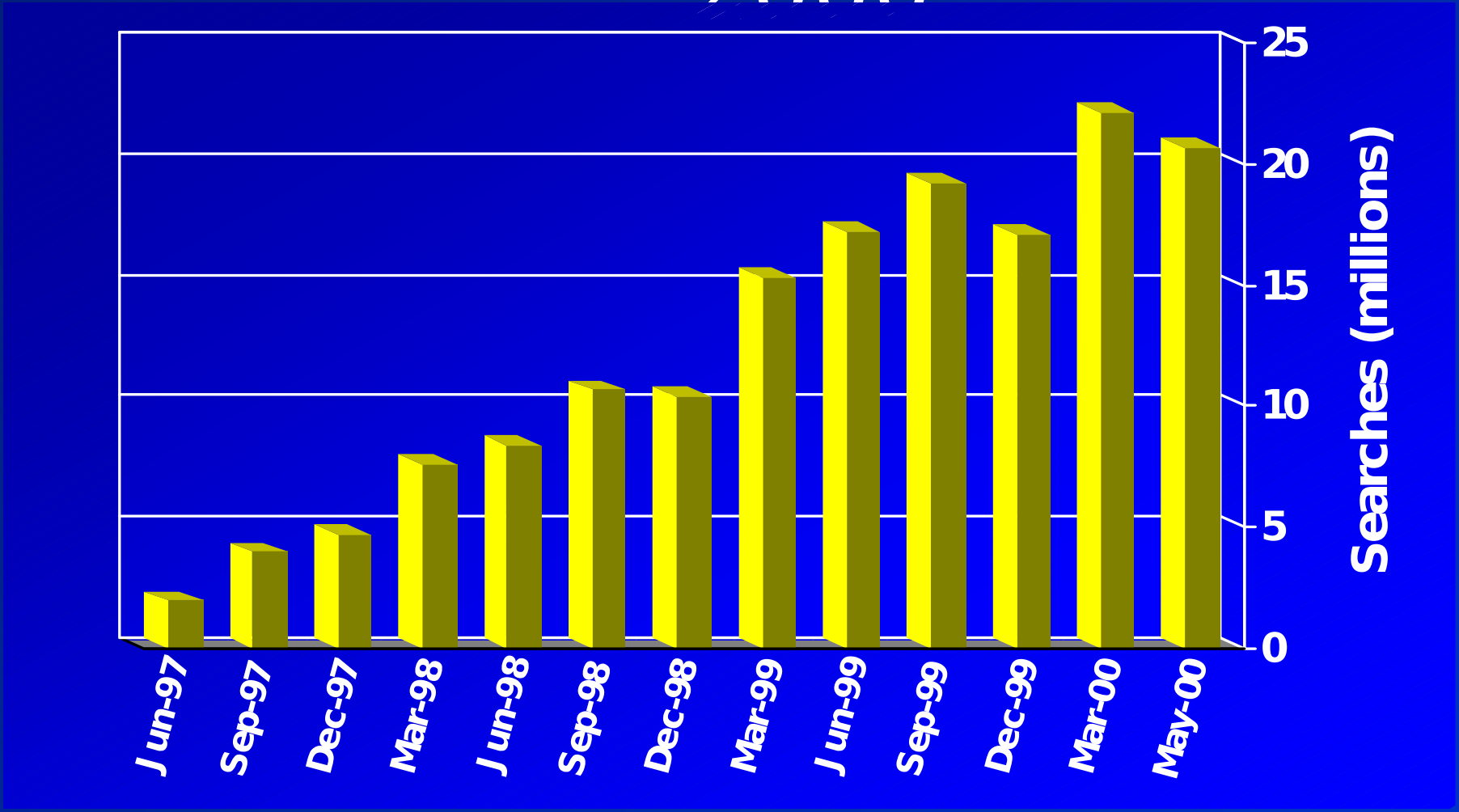

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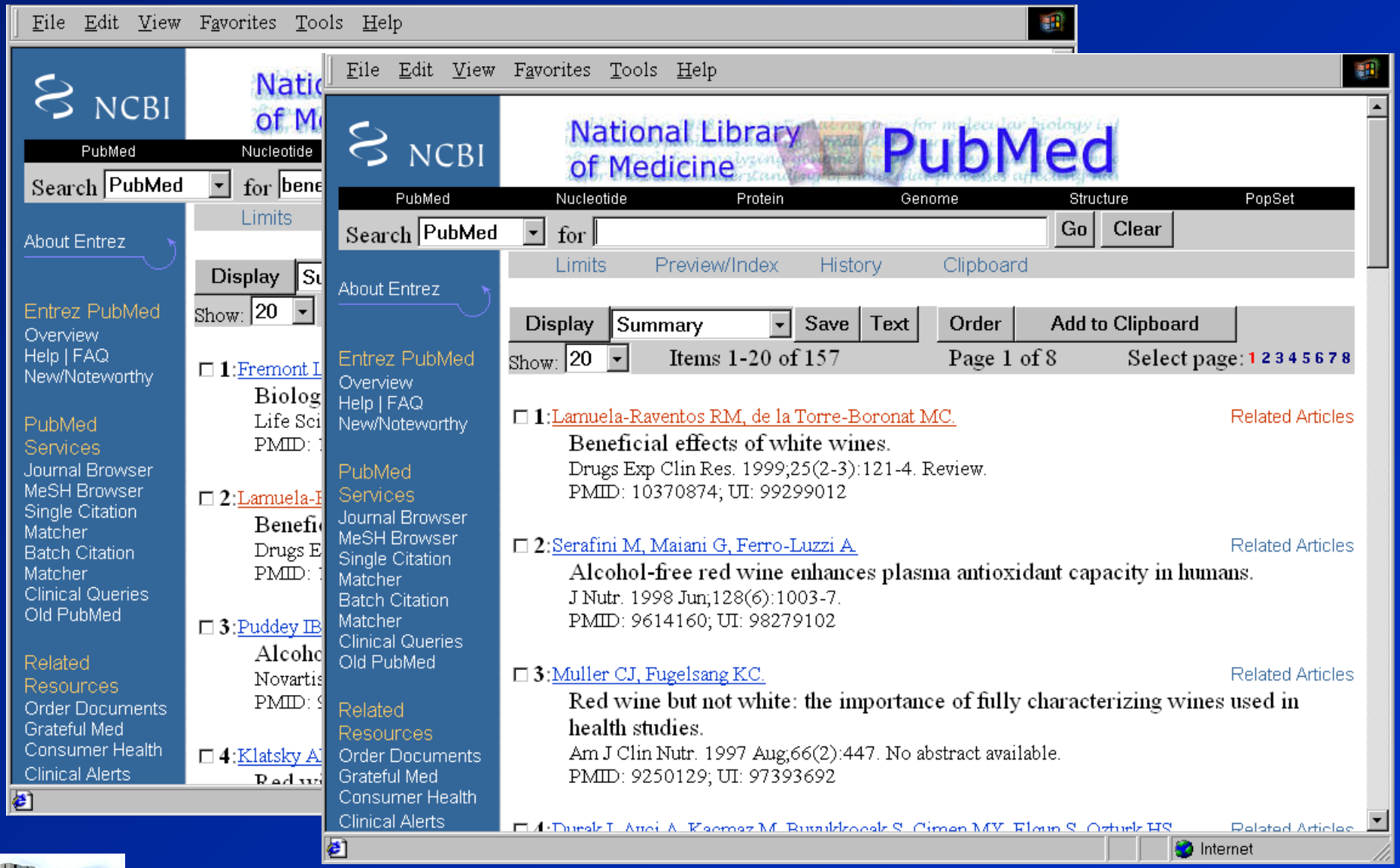

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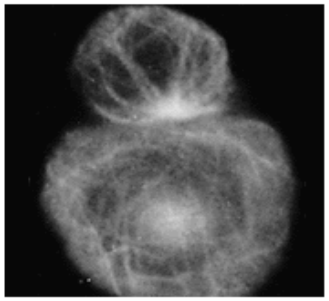
The Immune System

Cytotoxic T Cells



Outline

Introduction

Cytotoxic T Cells Recognize Fragments of Viral Proteins on the Surface of Virus-infected Cells

MHC-encoded ABC Transporters Transfer Peptide Fragments from the Cytosol to the ER Lumen

Cytotoxic T Cells Induce Infected Target Cells to Kill Themselves

Summary

Figures

Figure 23-52: Cytotoxic T cells in the process of killing target cells in culture

References

Schaerer, E.; Tschopp, J.Cytotoxic T cells keep their secrets. *Curr. Biol.* 3:167-169, 1993

Taylor, M.K.; Cohen, J.J.Cell-mediated cytotoxicity. *Curr. Opin. Immunol.* 4:338-343, 1992 [PubMed]

Cytotoxic T Cells Induce Infected Target Cells to Kill Themselves[34]

Once a cytotoxic T cell has recognized a viral peptide bound to a class I MHC molecule on the surface of a target cell, its job is to destroy the cell before the virus that gave rise to the peptide can produce new viral particles that can escape from the infected cell. When a cytotoxic T cell, caught in the act of killing its target, is labeled with anti-tubulin antibodies, its centrosome is seen to be oriented toward the point of contact with the target cell (Figure 23-52). Moreover, antibody labeling shows that talin, a protein that helps link cell-surface receptors to cortical actin filaments, is concentrated in the cortex of the T cell at the contact site. It is thought that the aggregation of T cell receptors at the contact site leads to a local, talin-dependent alteration in the actin filaments in the cell cortex; a microtubule-dependent mechanism then moves the centrosome and its associated Golgi apparatus toward the contact site, focusing the killing machinery on the target cell. A similar cytoskeletal polarization is seen when a helper T cell functionally interacts with a target cell.

The mechanism by which cytotoxic T cells kill their targets is not known for certain. They seem to employ at least two strategies, both of which are thought to operate by inducing the target cell to undergo *programmed cell death* (also called *apoptosis*, see p. 1169). In one strategy, binding to a target cell stimulates these cytotoxic T cells to release pore-forming protein called perforin, which is homologous to the complement component C9 and polymerizes in the target cell plasma membrane to form transmembrane channels. Perforin is stored in secretory vesicles and is released by local exocytosis at the point of contact with the target cell. The secretory vesicles also contain serine proteases and other proteins, which are also thought to play a part in killing the target cell, perhaps, by entering the target cell through the perforin channels and inducing programmed cell death. The second strategy, by contrast, involves the cytotoxic T cell activating a receptor on the surface of the target cell, thereby signaling the target cell to undergo programmed cell death.

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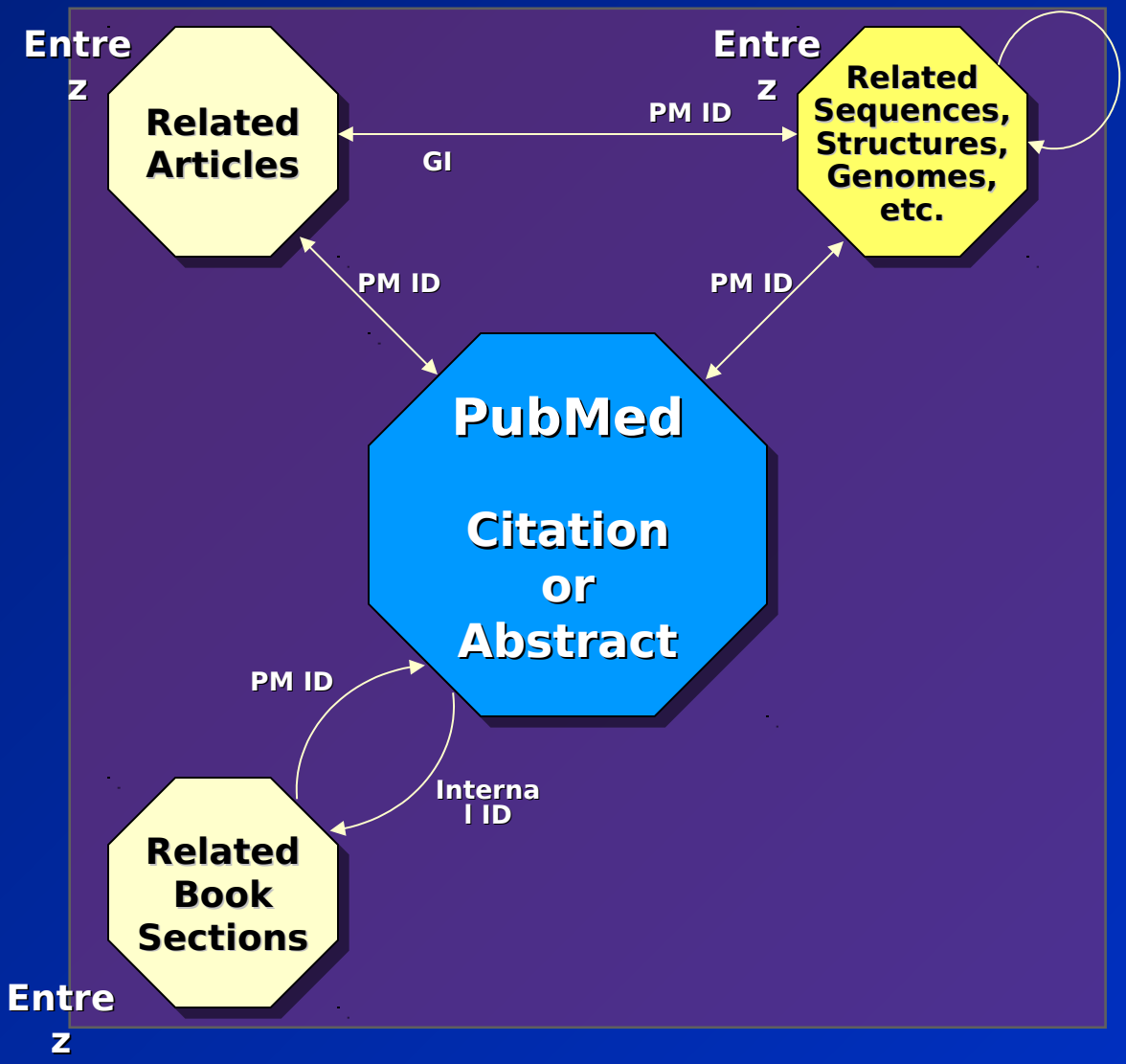

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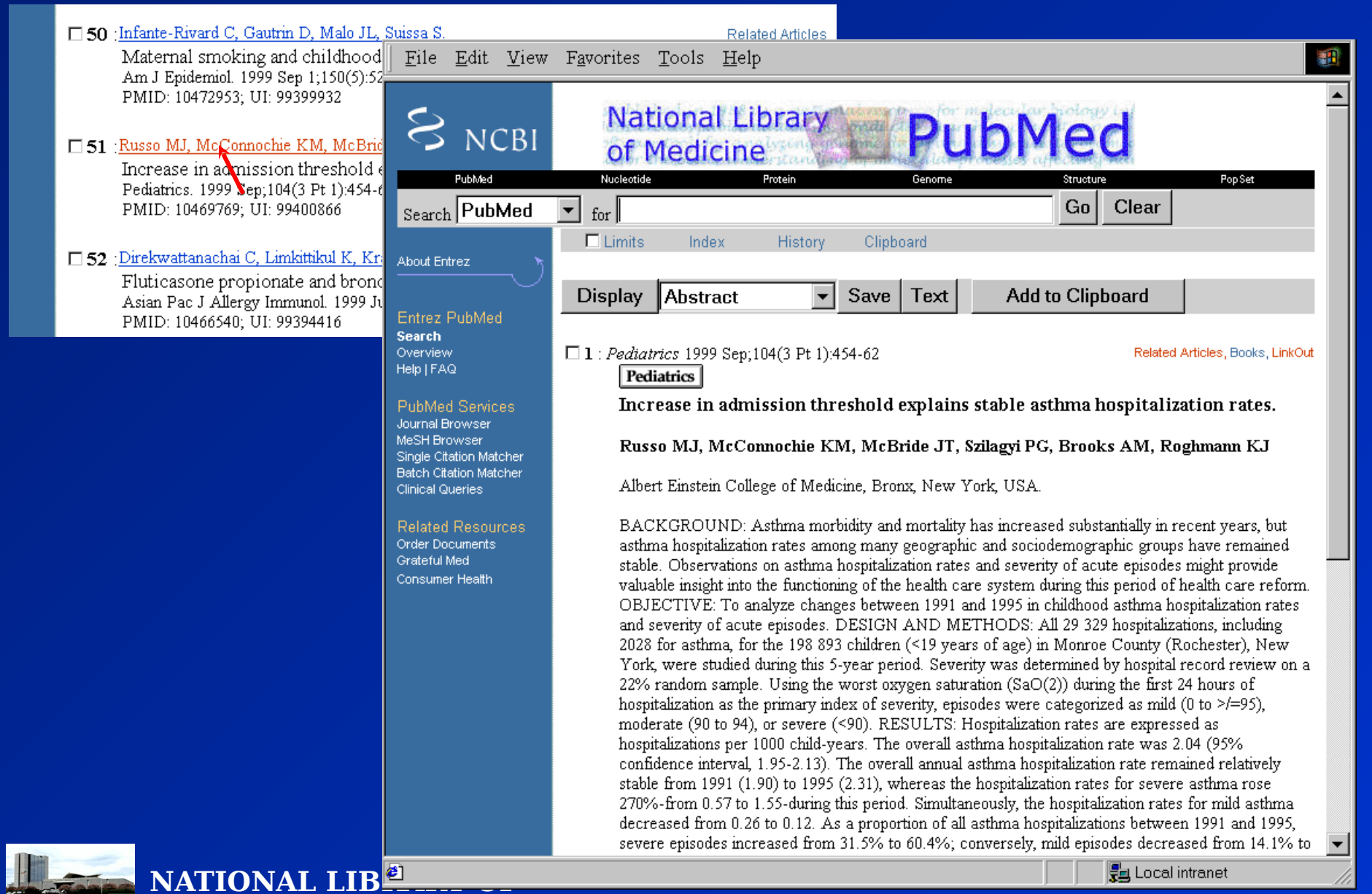
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Increase in admission threshold explains stable asthma hospitalization rates.
Russo MJ, McConnochie KM, McBride JT, Szilagyi PG, Brooks AM, Roghmann KJ
Albert Einstein College of Medicine, Bronx, New York, USA.
BACKGROUND: Asthma morbidity and mortality has increased substantially in recent years, but asthma hospitalization rates among many geographic and sociodemographic groups have remained stable. Observations on asthma hospitalization rates and severity of acute episodes might provide valuable insight into the functioning of the health care system during this period of health care reform. OBJECTIVE: To analyze changes between 1991 and 1995 in childhood asthma hospitalization rates and severity of acute episodes. DESIGN AND METHODS: All 29 329 hospitalizations, including 2028 for asthma, for the 198 893 children (<19 years of age) in Monroe County (Rochester), New York, were studied during this 5-year period. Severity was determined by hospital record review on a 22% random sample. Using the worst oxygen saturation (SaO(2)) during the first 24 hours of hospitalization as the primary index of severity, episodes were categorized as mild (0 to >/=95), moderate (90 to 94), or severe (<90). RESULTS: Hospitalization rates are expressed as hospitalizations per 1000 child-years. The overall asthma hospitalization rate was 2.04 (95% confidence interval, 1.95-2.13). The overall annual asthma hospitalization rate remained relatively stable from 1991 (1.90) to 1995 (2.31), whereas the hospitalization rates for severe asthma rose 270%-from 0.57 to 1.55-during this period. Simultaneously, the hospitalization rates for mild asthma decreased from 0.26 to 0.12. As a proportion of all asthma hospitalizations between 1991 and 1995, severe episodes increased from 31.5% to 60.4%; conversely, mild episodes decreased from 14.1% to
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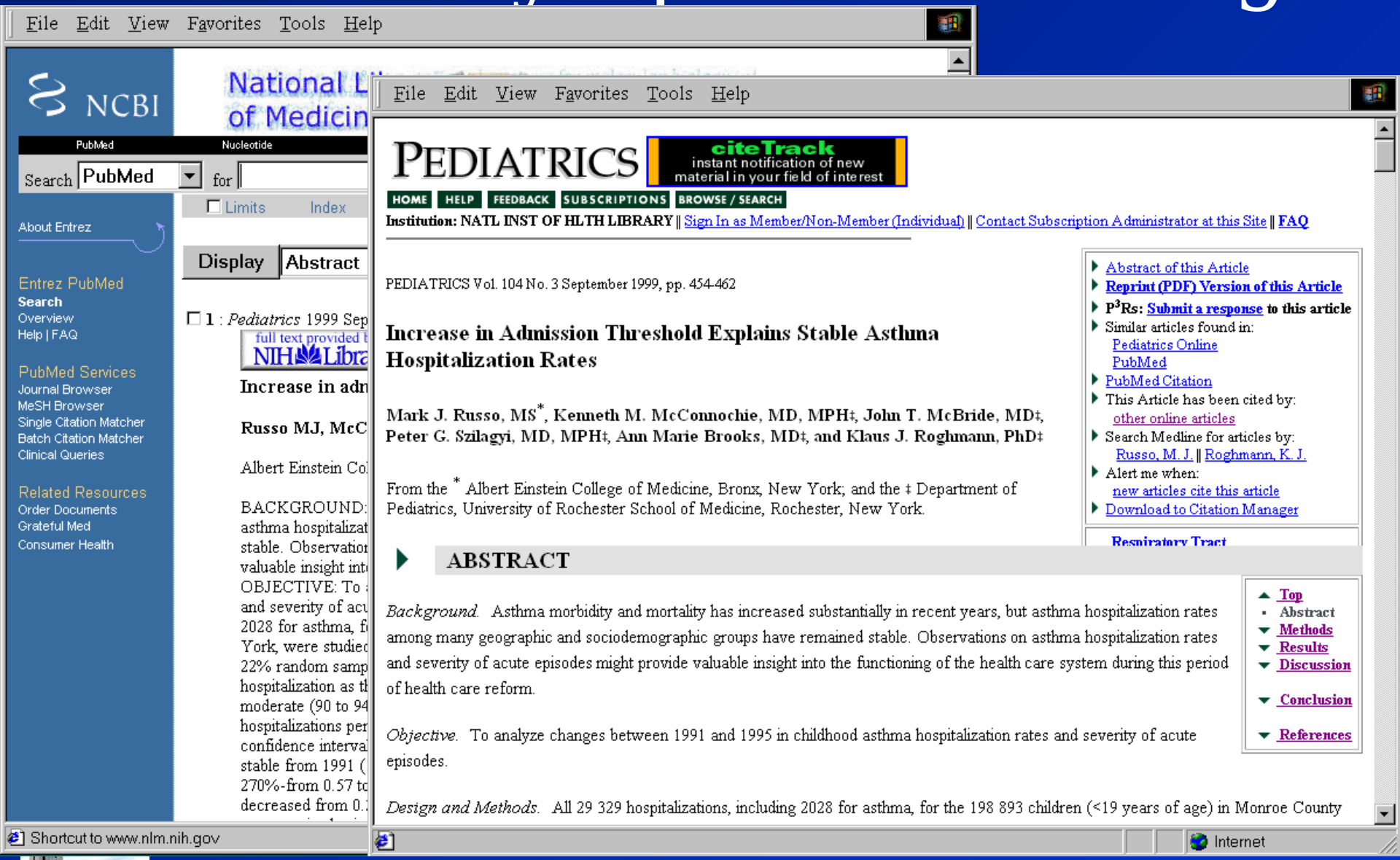
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PEDIATRICS Vol. 104 No. 3 September 1999, pp. 454-462
Increase in Admission Threshold Explains Stable Asthma Hospitalization Rates
Mark J. Russo, MS*, Kenneth M. McConnochie, MD, MPH‡, John T. McBride, MD‡, Peter G. Szilagyi, MD, MPH‡, Ann Marie Brooks, MD‡, and Klaus J. Roghmann, PhD‡
From the * Albert Einstein College of Medicine, Bronx, New York; and the ‡ Department of Pediatrics, University of Rochester School of Medicine, Rochester, New York.
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Objective. To analyze changes between 1991 and 1995 in childhood asthma hospitalization rates and severity of acute episodes.
Design and Methods. All 29 329 hospitalizations, including 2028 for asthma, for the 198 893 children (<19 years of age) in Monroe County
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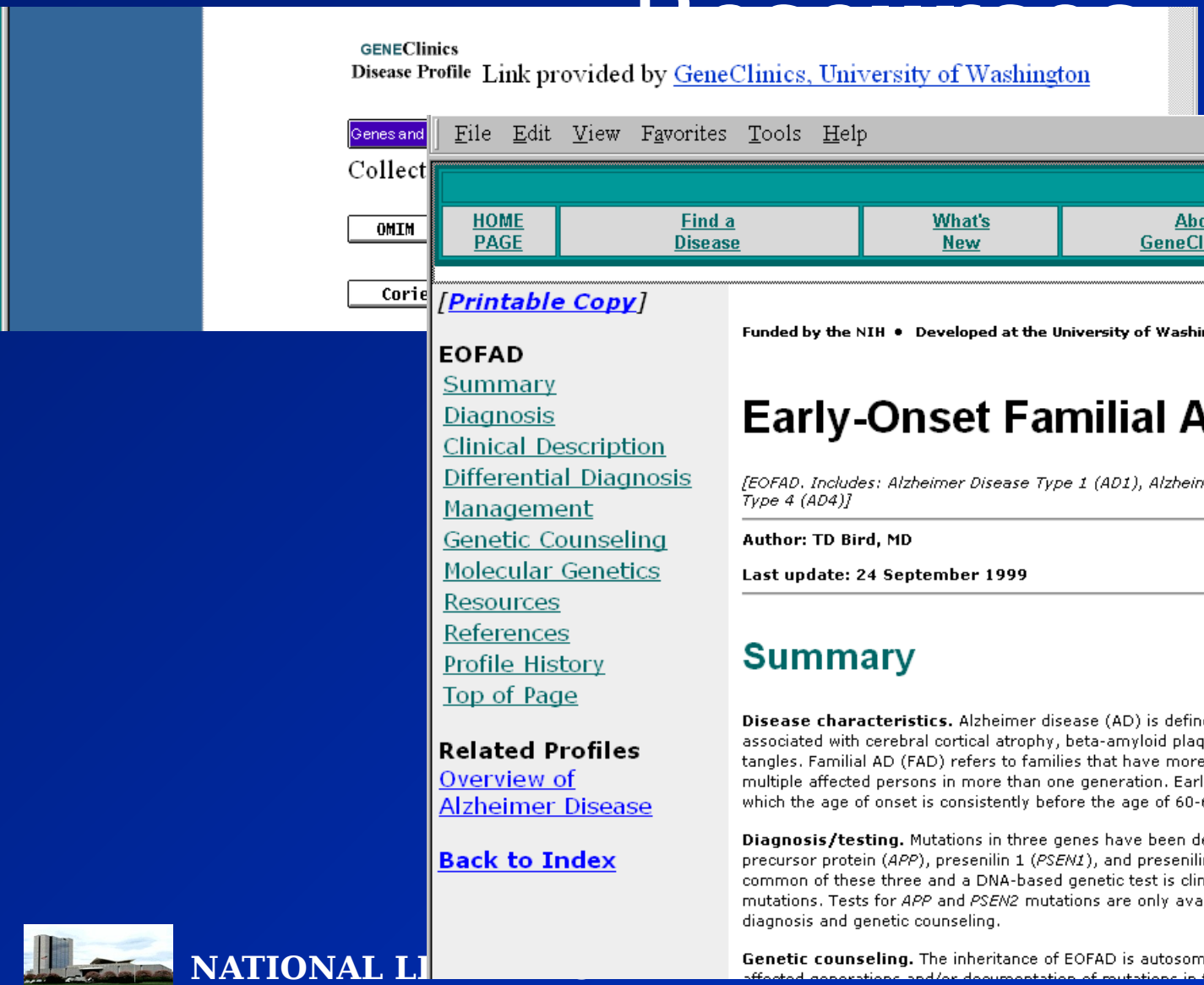
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GENEClinics
Early-Onset Familial Alzheimer Disease
[EOFAD. Includes: Alzheimer Disease Type 1 (AD1), Alzheimer Disease Type 3 (AD3), Alzheimer Disease Type 4 (AD4)]
Author: TD Bird, MD
Last update: 24 September 1999
Summary
Disease characteristics. Alzheimer disease (AD) is defined as adult-onset progressive dementia associated with cerebral cortical atrophy, beta-amyloid plaque formation, and intraneuronal neurofibrillary tangles. Familial AD (FAD) refers to families that have more than one case of AD and usually implies multiple affected persons in more than one generation. Early-onset FAD (EOFAD) refers to those families in which the age of onset is consistently before the age of 60-65 years and often before age 55.
Diagnosis/testing. Mutations in three genes have been demonstrated to cause EOFAD: amyloid precursor protein (APP), presenilin 1 (PSEN1), and presenilin 2 (PSEN2). Mutations in PSEN1 are the most common of these three and a DNA-based genetic test is clinically/commercially available for PSEN1 mutations. Tests for APP and PSEN2 mutations are only available on a research basis. Testing is used in diagnosis and genetic counseling.
Genetic counseling. The inheritance of EOFAD is autosomal dominant in those families with multiple

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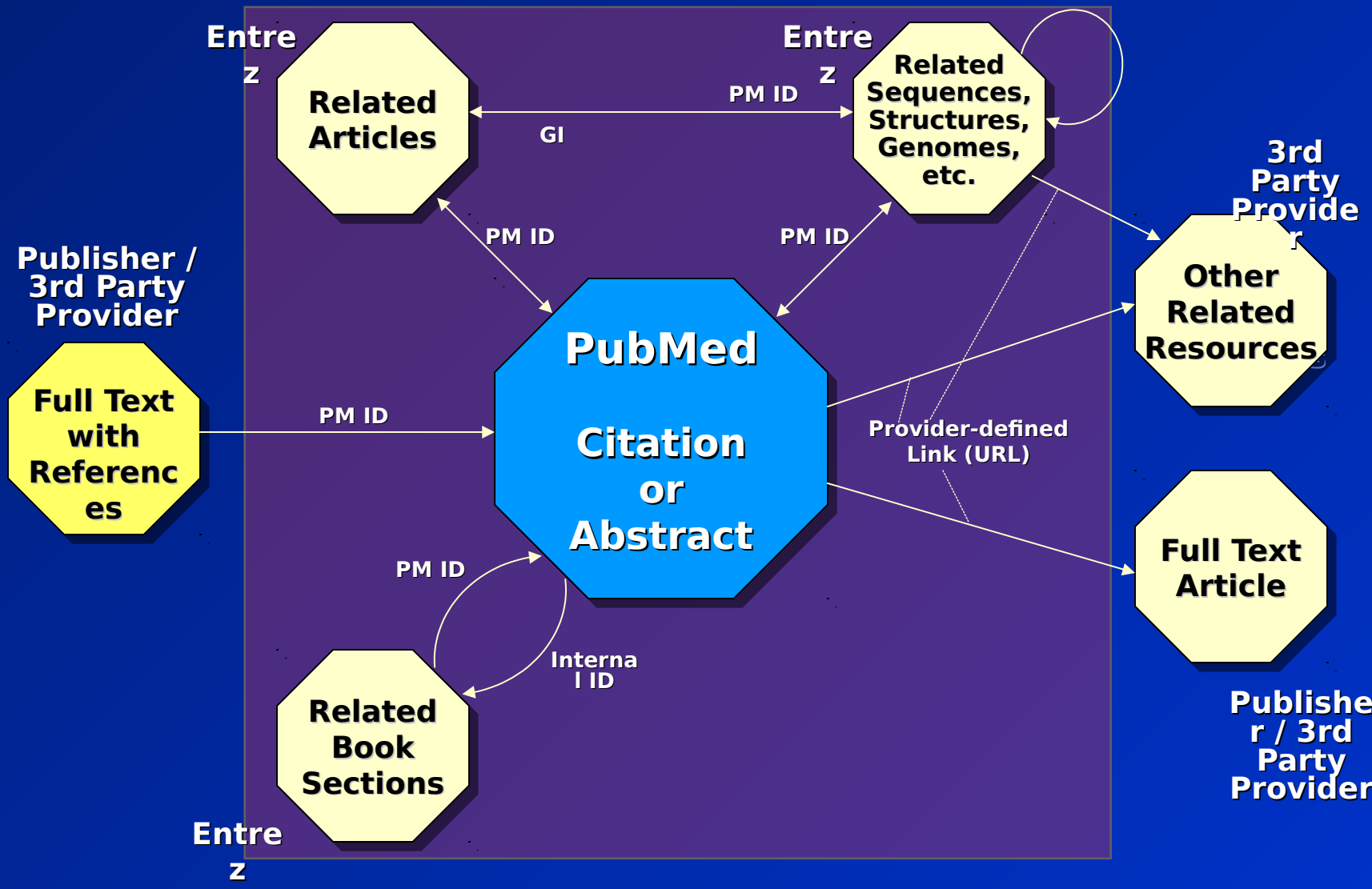

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An Endosome-to-Plasma Membrane Pathway Involved in Trafficking of a Mutant Plasma Membrane ATPase in Yeast

Wen-jie Luo and Amy Chang*

Department of Anatomy and Structural Biology, Albert Einstein College of Medicine, Bronx, New York 10461

Submitted June 21, 1999; Revised October 5, 1999; Accepted November 15, 1999

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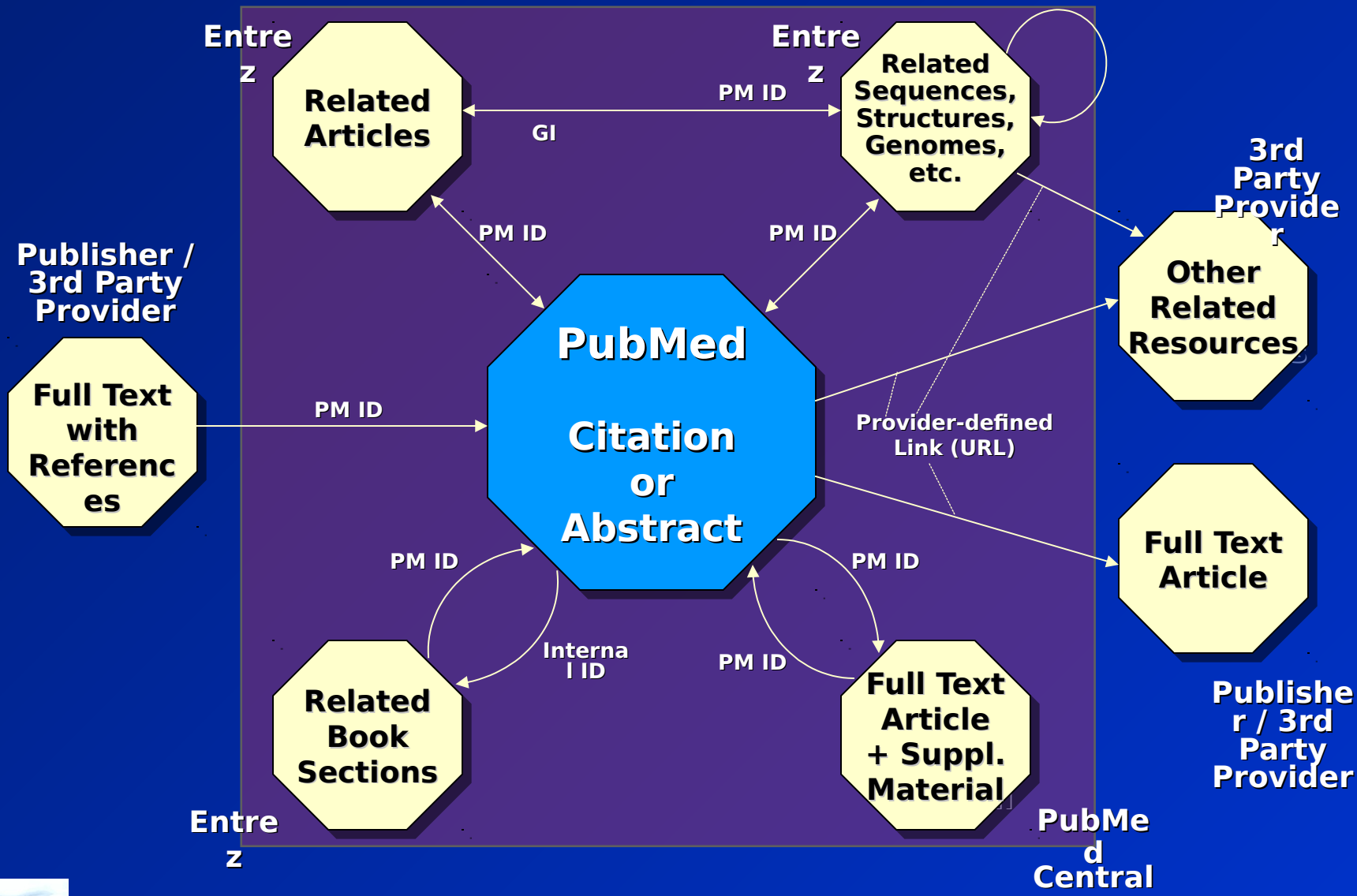

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